STRUCTURAL ELEMENTS IN THE CONCEPT OF MOTION SICKNESS

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SUMMARY PAGE

A slow rotation room in a laboratory environment provides an excellent instrument for the study of motion sickness because the experimenter can control not only the stressful Coriolis accelerations, but also other important procedural and environmental variables. By exploiting this control, combined with the judicious selection of experimental subjects, it was possible to confirm many previous findings and demonstrate that manifestations of disturbances in the vestibular system fall into two distinct categories. In the first category are reflex phenomena evoked by Coriolis accelerations when the head is rotated out of the plane of the room's rotation, and revealed through systems which, under natural stimulus conditions, have functional articulations with vestibular receiving areas. Included here are a characteristic sensation of tumbling or rotation, the Coriolis oculogyral illusion, nystagmus, dizziness, and neuromuscular incoordination. The symptomatology in the second category comprises an epiphenomenon superimposed on any manifestation of the first, when the unusual vestibular activity, presumably through facilitory-inhibitory processes, irradiates to cells or cell assemblies not normally stimulated.

These epiphenomenal responses are absurd in terms of the needs of the organism, and there is no evidence that their homeostatic mechanisms make any contribution toward restoring a homeostatic state in the vestibular system. The sites of origin of these responses are not vestibular receiving areas under normal circumstances, and the linkage with the vestibular system must be facultative. The relations between manifestations in the two categories suggest that the initial linkage, with certain sites at least, may be neuronal, but that chemical stimuli must be implicated to account for 1) long latencies between stimulus and response, and 2) the long perseveration of the manifestations after the stressful accelerations have ceased. Selected experimental findings are used in defining the characteristics of manifestations in the two categories and in demonstrating the nature of the facultative linkage between the otherwise independent systems underlying manifestations in the two categories. It will be shown that the experimenter, by manipulating mainly vestibular homeostatic mechanisms, can prevent the appearance of manifestations in the second category, control their severity when evoked, and lose control only when these symptoms are relatively severe or perseverate long after the stressful accelerations have ceased. Practical and theoretical implications are discussed, including the concept of "functional vestibular reserve."

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It is with pleasure that I acknowledge my debt to co-workers, and to Dr. Margaret Smith for the presentation in Figure 14 and for a critical reading of the manuscript.

INTRODUCTION

Since it is generally accepted that loss of function of the nonacoustic labyrinth confers immunity to motion sickness, we may with advantage compare the difference in behavior of the organism when the vestibular organs are stimulated under natural and under unnatural conditions. Under natural conditions, the behavioral responses to which the vestibular system* contributes are characterized by automaticity, reliability, and egality among members of a species or subspecies. There is little if any manifestation of "disturbance" in vestibular mechanisms under natural conditions, implying that the incredibly complex integrative mechanisms intercalated between sensory input and motor output have been effected in elegant fashion. Potentially, this "silent elegance" is of such importance to the organism that it could have evolved only through natural selection and survival of the fittest. This backward look at our evolutionary development is enormously important in appreciating the harmony which characterizes functions subserved by the vestibular organs under physiological conditions and our inherent limitations in adjusting to the environment when the vestibular organs are stimulated under unnatural conditions.

Nature's guarantee no longer holds good when the vestibular organs are stimulated either by exposing the organism in an unusual force environment or by some other artificial stimulation involving one or both of the paired organs. The vestibular system, delicately balanced right-left and with extensive articulations in the central nervous system, is easily disturbed. The disturbance may be manifested by abnormal behavioral responses which not only fall into two distinct categories but also have curious functional relationships. One category involves 1) the vestibular system and such other systems which under normal conditions receive vestibular activity, and 2) additional systems which may exert modulating influences. Until better terminology is devised, the vestibular responses in this first category will be termed V-I manifestations, their underlying systems V-I systems, and the stressful vestibular stimulations which disturb only this system, V-I stressors.

When the vestibular organs are stimulated under certain unnatural conditions, epiphenomena appear, characterized by manifestations whose sites of origin are not, under normal conditions, vestibular receiving areas. The manifestations in this second category, termed V-II manifestations, include not only the symptomatology of motion

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*Although it has been demonstrated not only that some of these responses have their genesis in the semicircular canals and others in the otolith apparatus, but also that the latter may modulate canalicular responses, nevertheless, our knowledge of the relationships between these two sensory organs is incomplete, and our purpose here is better served using the combining term labyrinthine or vestibular organs.

sickness as usually described, but also many other symptoms as well. The systems underlying V-II manifestations are termed V-II systems and the stressful stimuli, VII stressors.*

Although this is not the place to define "motion sickness," it is important at this time to point out significant similarities between this term and V-II manifestations. Motion sickness is a clinical diagnostic term implying certain criteria have been met to ensure validity. Thus, a close temporal order of exposure to "motion" generating stressful accelerations and the appearance either of vomiting or some combination of such cardinal symptoms as nausea, pallor, sweating, increased salivation, and drowsiness, constitutes, respectively, a pathognomonic or valid diagnosis. On the other hand, manifestations of the V-II category may be evoked by various means other than "motion" and, under proper laboratory conditions, a single V-II symptom in the symptom complex may be elicited and studied, as will be shown later on. The use of the term V-II avoids the constraints imposed by "motion sickness" and invites the experimenter to study all epiphenomenal responses which range over a wider spectrum than that defined as motion sickness. The question may be raised which symptoms are first, second, or third order or, if one prefers, "complications." For example, pallor, which may be preceded by flushing, is mainly due to constriction of capillaries and, to a lesser degree, by arterioles. It is then a second-order symptom. The electrolytic imbalance following vomiting, which is certainly consequent to other earlier initial disturbances, might persist as a "complication" for a relatively long period after other V-II manifestations have disappeared.

When the conceptual structure elucidating the mechanisms *underlying* V-II manifestations is finally determined, it is safe to venture the opinion that the keystone will be represented by the facultative linkages between disturbances in V-I and V-II systems. In Figure 1 is shown a highly simplified scheme indicating events subsequent to thermal stimulation of the labyrinth. A nonaccelerative stimulus is chosen here, partly to exemplify the general application of the concept to be developed and partly because the effects of Coriolis accelerations will be depicted later. In order to avoid an unmanageable number of variables, the term "secondary influences" is used to include the role of 1) nonvestibular sensory inputs, 2) central commands, and 3) all other modulating influences affecting V-I and V-II systems. Note in Figure 1 that a very weak threshold stimulus may not involve the facultative linkage but that a somewhat stronger stimulus might, to some extent. In a susceptible person vomiting may occur subsequent to bilateral irrigations of contrasting temperatures, followed by disturbances in electrolytic balance which might last for more than 24 hours before there is a return to normal.

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*The term "V-II stressor" will imply that it is also a V-I stressor. Occasionally, the terms V-I and V-II stressors are used to indicate their different roles.

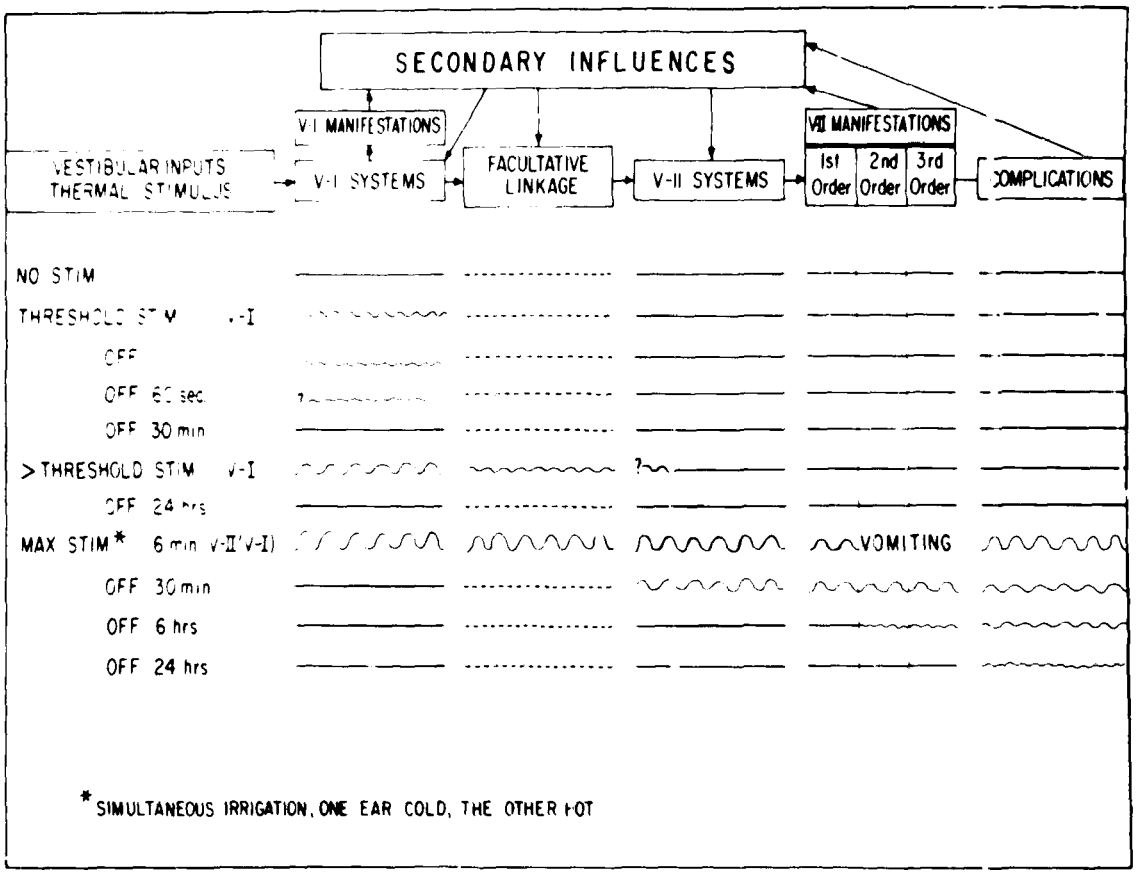

Figure 1

Schema depicting possible events and processes underlying manifestations evoked following thermal stimulation of nonacoustic labyrinth. Solid lines: no manifestations; no disturbances in systems. Wavy lines: size indicates magnitude of above events. Facultative linkage: dotted lines, no crossing; wavy lines, intensity of vestibular irradiation.

Whenever advantageous in the discussion to follow, use will be made of the concept and terminology of homeostasis. Since its introduction, Cannon's concept has been broadened to include all events and processes in living systems at any or all organizational levels involved in adjusting to the environment.

Nearly all of the findings used in this presentation were made in slow rotation rooms at the Naval Aerospace Medical Institute by various members of the staff. These will be drawn upon in preference to any similar observations made elsewhere, partly because of our greater familiarity with them and partly because of the similarity of environmental conditions under which they were obtained.

THE SLOW ROTATION ROOM (SRR)

A slow rotation room in a laboratory setting constitutes a powerful instrument for the study of experimental motion sickness, and some knowledge of its characteristics is essential to the understanding of what follows. The two slow rotation rooms at the Naval Aerospace Medical Institute resemble fully enclosed carousels, one about 14 and the other 20 feet in diameter, provided with housekeeping facilities, laboratory equipment, and communication systems. In the SRR a person is not subjected to a stressful stimulus unless he rotates his head out of the plane of the room's rotation; hence, the situation differs from that in ships and planes where a person cannot avoid stressful accelerations generated by motions of the vehicle. The cardinal advantages of such a room lie in 1) its habitability, 2) the great range in strength of the disturbing stimulus, 3) the experimenter's control over the stressful accelerations, and 4) the opportunity for measurements to be made with sophisticated laboratory equipment. Three important factors contribute to habitability. First is the fact that very little more effort is required to move the head and generate the maximal stressful stimulus than is the case when the room is stationary. Second, at constant velocity there is little or no awareness that the room is rotating. The third factor concerns the adequate housekeeping facilities, food, and recreation. Habitability has been demonstrated by the fact that subjects have remained in the SRR for over a month with experimenters in attendance.

It is important to emphasize the great range in strength of the disturbing stimulus to the labyrinth. At 1.0 rpm there is little or no manifest disturbance of vestibular origin, whereas, at 10.0 rpm it is comparable to exposure on rough seas. Control over the stressful Coriolis accelerations involves regulation of the room's angular velocity and of the head rotations made by the subject. By standardizing the "head rotations" or the general activity "tasks" involving head rotations, the strength of stressful stimulus becomes a function of the room's angular velocity. At constant velocity, rotation of the head can be controlled as to number of planes, degree of arc, periodicity, and total number. Moreover, the stressful stimulus ends quite soon after the end of the head rotation. Coriolis accelerations can be manipulated with greater ease, precision, and amount than is true of any other type of acceleration stress subserving the same purposes. Moreover, there is an incremental increase in severity of

the stressor effect with increasing strength of Coriolis acceleration force. The experimenter's control is limited only by the inability of the subject to continually make the standardized head motions, and when they cease, the stressor is "off" and recovery sets in.

Through choice of subjects and control over environmental factors in the SRR the experimenter can exercise important options in evoking symptoms which differ in kind and severity. Moreover, from a study of the symptomatology, such derived phenomena as 1) latency, 2) temporal summation or "cumulation," 3) temporal perseveration, 4) adaptation, 5) transfer effects, and 6) conditioning will throw light on the mechanisms underlying the manifestations.

CATEGORIES OF MANIFESTATIONS

CATEGORY I (V-I)

This category embraces all manifestations resulting from disturbances of the V-I system, and systems with which it normally articulates. It specifically does not include V-II manifestations which result from a crossover of vestibular activity (or influence) to cell assemblies of another system with which, normally, the V-I system does not articulate. Although the identical stressful stimulus may cross the facultative linkage between V-I and V-II systems, depending on secondary influences and whether adaptation within the V-I system has occurred, the need for differentiating between a stimulus which does or does not cross at a particular point in time is met by the terms, V-I and V-II stressors.

When persons who have normal function of the nonacoustic labyrinth, yet are highly insusceptible to motion sickness, are exposed to moderately stressful Coriolis accelerations in the SRR, V-I manifestations appear in the absence of overt V-II symptoms. The discussion to follow will be limited to five symptoms which typify the effects of Coriolis but not necessarily those of other unusual accelerations on the vestibular system.

A highly characteristic "sensation" (22, 44) of a complex "rotation" or "tumbling" is experienced when moving the head out of the plane of the room's rotation, and this sensation may persist for a few seconds at the end of the movement, due, presumably, in large part to inertial lag of the endolymph. This sensation is maximal initially and is gradually lost after a large number of such head movements are made. The strength and duration of this sensation depend partly on whether the rotation of the head is with or against the direction of the room's rotation and whether the motion is "away from" or "return to" the upright. Much individual variance has been demonstrated regarding susceptibility, but this sensation is experienced by all persons with normal labyrinths.

A form of visual apparent motion termed the Coriolis or Coriolis oculogyral illusion (15) is readily perceived on flexing the head away from the upright. This apparent motion is maximal during initial head rotations and disappears after repeated

head motions. The illusion tends to be suppressed in a lighted room and is far more prominent when a dimly lighted target is viewed in darkness; then it may be readily perceived during, and for a very brief period after cessation of, rotation. There is very great individual variance with regard to the number of head movements required to reduce greatly or extinguish this illusion.

Coriolis nystagmus is readily demonstrated by means of nystagmography under the same stimulus conditions required for the perception of the Coriolis oculogyral illusion, although there is an advantage in having the eyes closed. Indeed, the behavioral characteristics of the two phenomena are similar and have been studied in some detail.

The findings in one of these experiments (21) are relevant to this discussion, although only about one third of the subjects were free from symptoms of motion sickness. The subjects were seated in the SRR throughout this experiment and were immobile except for a series of measured head rotations restricted to the frontal plane and to a 45° quadrant of that plane with the room rotating at 7.5 rpm. The Coriolis illusion (Figure 2) and nystagmus (Figure 3) were reduced or abolished after a sufficient number of head rotations, but this adaptation did not transfer to the "unpracticed" quadrant of the same plane, at least to any considerable degree. Rotation of the head in the unpracticed direction evoked responses similar to those manifested at the onset of rotation.

On cessation of rotation, movement of the head in the practiced direction, in some subjects at least, evoked the illusion and nystagmus, both of which were in the opposite sense to those manifested at the onset of rotation. These findings suggested that the mechanism underlying the adaptation was in the nature of a compensatory mechanism of opposite sign and raised the question whether they might be conditioned responses, inasmuch as the illusion and nystagmus were evoked in a stationary environment in the absence of a Coriolis acceleration.

Dizziness is a sometime "complaint" of a minority of subjects. It is usually mild and either more likely or more prominent with eyes open than closed. There is some uncertainty whether it represents a first- or second-order symptom, but in either event it soon disappears after practice in making head movements.

Motor incoordination may be demonstrated when a subject carries out a psychomotor task while being passively rotated away from the upright, and it is reasonable to assume that the genesis is mainly in disturbances in the vestibular system. With active movements of the upper part of the body from the waist, notably forward and return to the upright, the tendency to move in a slightly curved plane suggests the effect of a Coriolis force acting on the body. The fact that this tendency is manifested by labyrinthine-defective (L-D) subjects proves that forces not acting on the vestibular organs exert an influence.

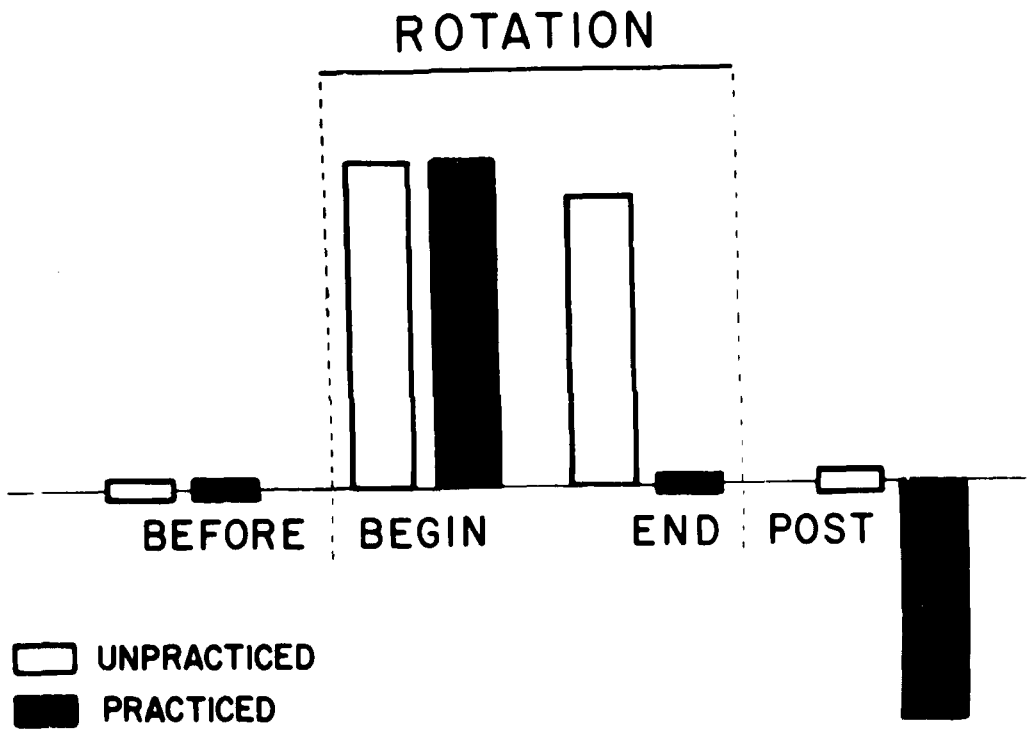

Figure 2

Comparative magnitude and direction of the Coriolis illusion associated with single head movements before, during, and after prolonged rotation at 5.4 rpm. Tests carried out at 7.5 rpm.

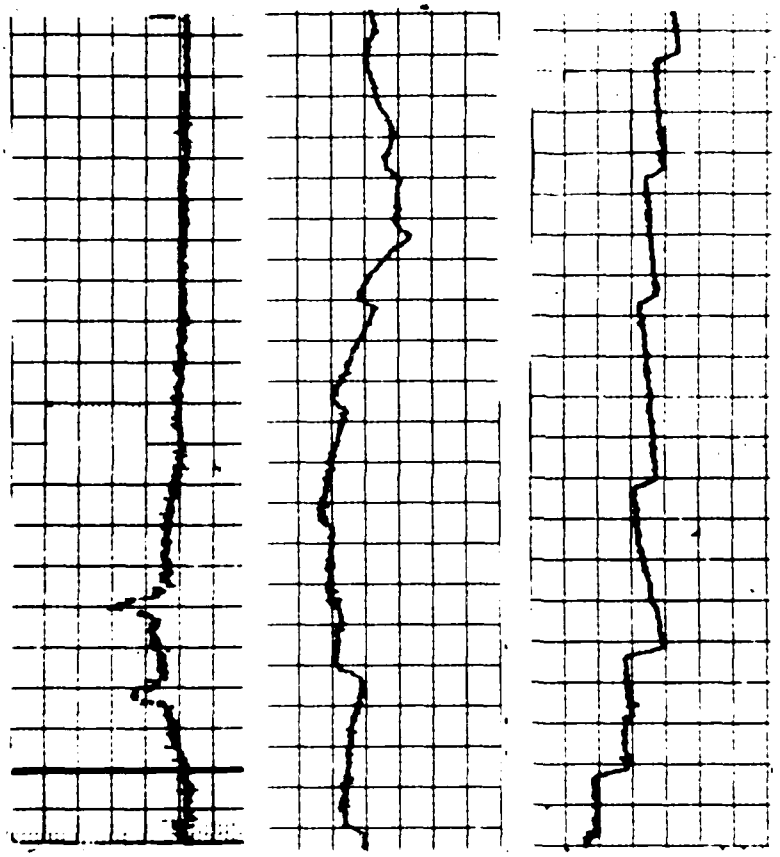

Figure 3

Nystagmograms obtained from a healthy subject while flexing the head toward the shoulders: (1) before, (2) at the onset of prolonged rotation in the SRR, and (3) after cessation of rotation. Note change in direction of beat between 2 and 3.

Ataxia

The ataxia manifested in the rotating room resembles that experienced aboard ship, and it is possible to demonstrate the contributing role of the vestibular organs by comparing the responses of normal and of L-D subjects. With the onset of rotation, both normal and L-D subjects experience difficulty in walking which is maximal initially and becomes progressively less over a period of days, after which there is little further change. This may be shown by a test for postural disequilibrium designed to reveal small differences between the normal and L-D subjects in a stationary environment. One significant difference between the normal and L-D subject is that the former, on sudden movement of the head, is more disturbed in his postural equilibrium than is the L-D subject. On cessation of rotation both normal and L-D subjects manifest ataxia on walking. The sensations differ from those experienced on disembarking after a sea voyage in that the subjects report that they feel unstable on a stable platform, whereas after a voyage the platform seems to be unstable too. Again, the normal subject on quickly rotating the head, experiences disequilibrium and may experience dizziness; these are not experienced by the L-D subject.

Comments

The typical V-I manifestations just described, and due to vestibular disturbances resulting from Coriolis accelerations generated by rotations of the head out of the plane of the room's rotation, have the following in common: 1) short latencies characteristic of reflex phenomena, 2) maximal response to the initial stimulus, 3) modulation by secondary influences, 4) little or no evidence of temporal perseveration after the stressor is off, taking account of the inertia of the endolymph, 5) variable time-course of adaptation, and 6) need for readjustment on return to a stationary environment.

With repeated exposure to the stressor, homeostatic processes, striving to bring about a new stable state, are effective in so doing. An example suggested that the generation of a compensatory mechanism of opposite sign is involved in the establishment of a new homeostatic state.

The change from a rotating to a stationary environment again caused V-I disturbances on rotation of the head. Important implications include: 1) only rotation of the head is required to evoke manifestations; 2) the stimulus is "normal"; 3) some of the manifestations tend to be in the opposite sense to those experienced with the onset of rotation; and 4) holding the head fixed with respect to the Earth tends to prevent, and rotating the head tends to accelerate, adjustment to the stationary environment. Groen (19) has suggested that adaptation involves the "build up" of a pattern in a "center," either in the vestibular nuclei or elsewhere. At all events, only the vestibular system appears to be involved, with the possible exception of a conditioned stimulus mentioned earlier in connection with the fact that after cessation of rotation, normal head movements evoke symptoms. The explanation, however, may be that a normal stimulus and even a normal vestibular input would encounter an abnormal integrative pattern necessitating a change.

Some V-I manifestations which are readily demonstrated in a rotating room were not described inasmuch as they are not essential to our argument, such as the oculogravic illusion (9), and the typical oculogyral illusion (11) and associated nystagmus. These manifestations have some characteristics which differ from those just described but are just as readily recognized as V-I symptoms.

CATEGORY II (V-II)

The symptoms now to be described constitute an important example of "system cross-over manifestations"; i.e., they have their origin in two normally independent systems but are the manifestations of one. As seen in Figure 4, the first or initiating system is vestibular which stimulates the effector systems that evoke behavioral responses only after crossing a facultative linkage. The "once-removed" origin of V-II manifestations is sufficient to ensure that they are absurd in terms of vestibular mechanisms and nonsensical in terms of the human economy. The initial responses implicate the visceral nervous system. Instances will be demonstrated wherein the response seemed to be not only appropriate to the site of origin, but also a response to a "normal" stimulus. More commonly the responses, although in some ways appropriate, are deemed to have abnormal characteristics.

Experimental Findings

Rapid Increase to Terminal Velocity of the Slow Rotation Room with Subjects Engaged in General Activities During Prolonged Exposure Between 10.0 and 1.0 rpm. In our experience, without exception, normal persons became "sick" and L-D subjects were immune to V-II manifestations while carrying out assigned tasks and housekeeping activities at 10.0 rpm (13,18,25,32). Only the fact that the normal subjects restricted their head movements made the experience acceptable to them. The longest exposure was about 12 days (18), at the end of which the four young officer subjects had not regained their prerotation state of fitness. In addition to V-I manifestations, there are changes in respiration, minor cardiovascular alterations and gastrointestinal disturbances. There is confirmation of urinary excretion of an antidiuretic agent (48). Biochemical measurements reveal disturbances in electrolytic balance, glucose metabolism, and in the urinary excretion of the so-called stress hormones, 17-hydroxycorticosteroids, epinephrine, and norepinephrine. Hematological changes are observed consequent to disturbances in water balance, and there is a decrease in circulating eosinophiles as another evidence of exposure to stress. Occasionally a subject may fail to participate in a test for reasons of feeling ill, but otherwise performance measurements, with subjects seated and head fixed, reveal little or no decrement. Ataxia, severe at first, gradually becomes less troublesome and after three or four days there is little change in performance.

Generally, there is much individual variance in susceptibility and in the time-course of adaptation. In a given individual, following the onset of rotation, there is a gradual increase in the severity of symptoms. First-order effects give rise to secondary manifestations of still greater diversity involving major organs and systems and are

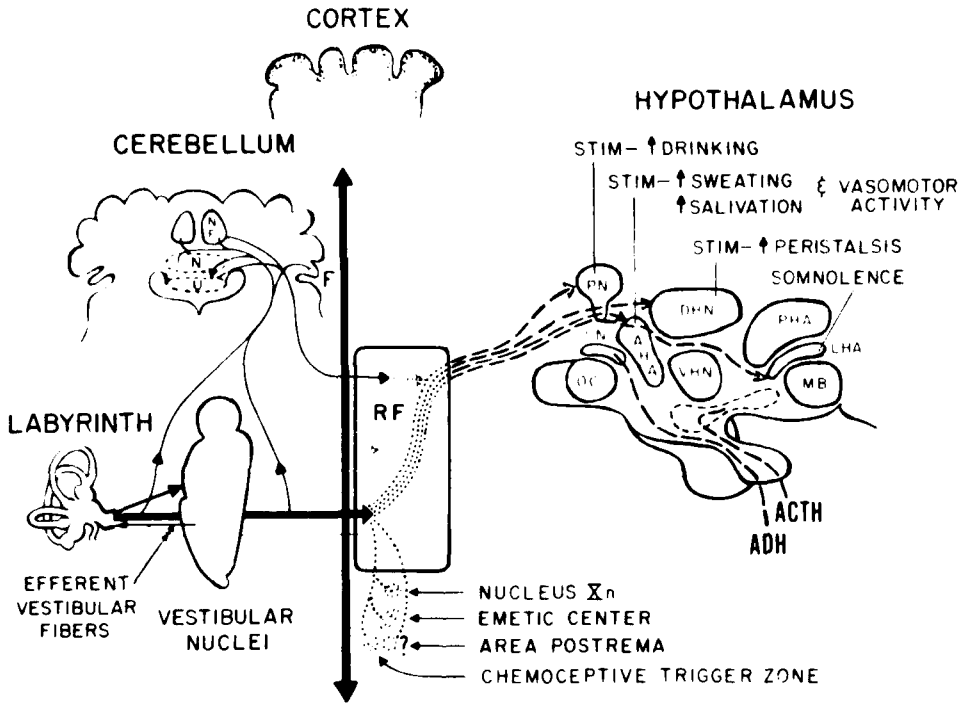

Figure 4

Depicting the possible (abnormal) modification of vestibular activity (dotted lines) following certain Coriolis acceleration

NF: Fastigial nucleus
U: Uvula
N: Nodulus
F: Flocculus
RF: Reticular formation
PN: Paraventricular nucleus
SN: Supraoptic nucleus
OC: Optic chiasma
AHA: Anterior hypothalamic area
DHN: Dorsomedial hypothalamic nucleus
VHN: Ventromedial hypothalamic nucleus
PHA: Posterior hypothalamic area
LHA: Lateral hypothalamic area
MB: Mammillary body

declared by subjective symptom and objective sign. In this chaotic state the experimenter is at a loss to do more than make serial appraisals and measurements which reveal the waxing and waning of specific responses, but little of the incredibly complex underlying mechanisms. The stressful stimulus leading to adaptation in the initiating V-I system is in competition with the stressor effect in the effector V-II system. Moreover, once severe symptoms have been evoked, they may, in turn, act as secondary influences causing an exacerbation of the symptoms.

Between the extremes of 10.0 and 1.0 rpm it was found that, for a given level of general activity, the two important factors governing the appearance of symptoms were the susceptibility of the unprotected subject and the angular velocity of the room. At 6.4 and 5.4 rpm even relatively unsusceptible subjects were handicapped until adapted to the nausea syndrome, but they were able to carry out all of the experimental tasks. At velocities of 3.82 to 1.71 there was a decreasing tendency for symptoms to appear, but those persons with average susceptibility readily adapted, and some difficulty in walking was a principal manifestation. A major experiment was conducted at 3.0 rpm in which three subjects and one experimenter were exposed to continual rotation for two weeks (25). The principal finding was the absence of any incapacitating disturbance of a physiological or psychological nature, either during or after cessation of rotation.

At 1.0 rpm (32) even highly susceptible subjects exposed in the SRR experienced trivial symptoms at the onset which were not aggravated as the result of experimenter-paced head motions. Despite the absence of overt V-I and V-II symptoms, the V-I system must be "disturbed" because of the fact that adaptation takes place.

Prevention of V-II Symptoms by Means of Incremental Increases to a Terminal Velocity of 10.0 rpm. Three attempts to prevent symptoms by step increases to a terminal velocity of 10.0 rpm were unsuccessful; two involved three incremental steps over a period of approximately three days, and the third a series of 40 incremental steps over a period of 40 hours (5).

In the next attempt, overt V-II manifestations, with the probable exception of drowsiness, were prevented solely by nine stepwise increases over a period of 16 days to a terminal velocity of 10.0 rpm (14). The stress profile and the symptomatology are summarized in Figure 5. There was no attempt to minimize head motions; on the contrary, the subjects had a busy schedule and at times made experimenter-paced head motions. With the exception of drowsiness, all other V-II symptoms were either trivial or explicable (due to power failure) in the perrotation period. Daily clinical evaluations by the onboard physician-experimenter, bolstered by routine hematological procedures, urinalysis, and other laboratory tests, revealed no definite variations from control values. The results of the analysis of the excretion rates for epinephrine and 17-hydroxycorticosteroids revealed no significant differences from baseline rates throughout the entire experimental period. On cessation of rotation, ataxia was the most prominent and lasting complaint, and symptoms of motion sickness were either absent or of small significance.

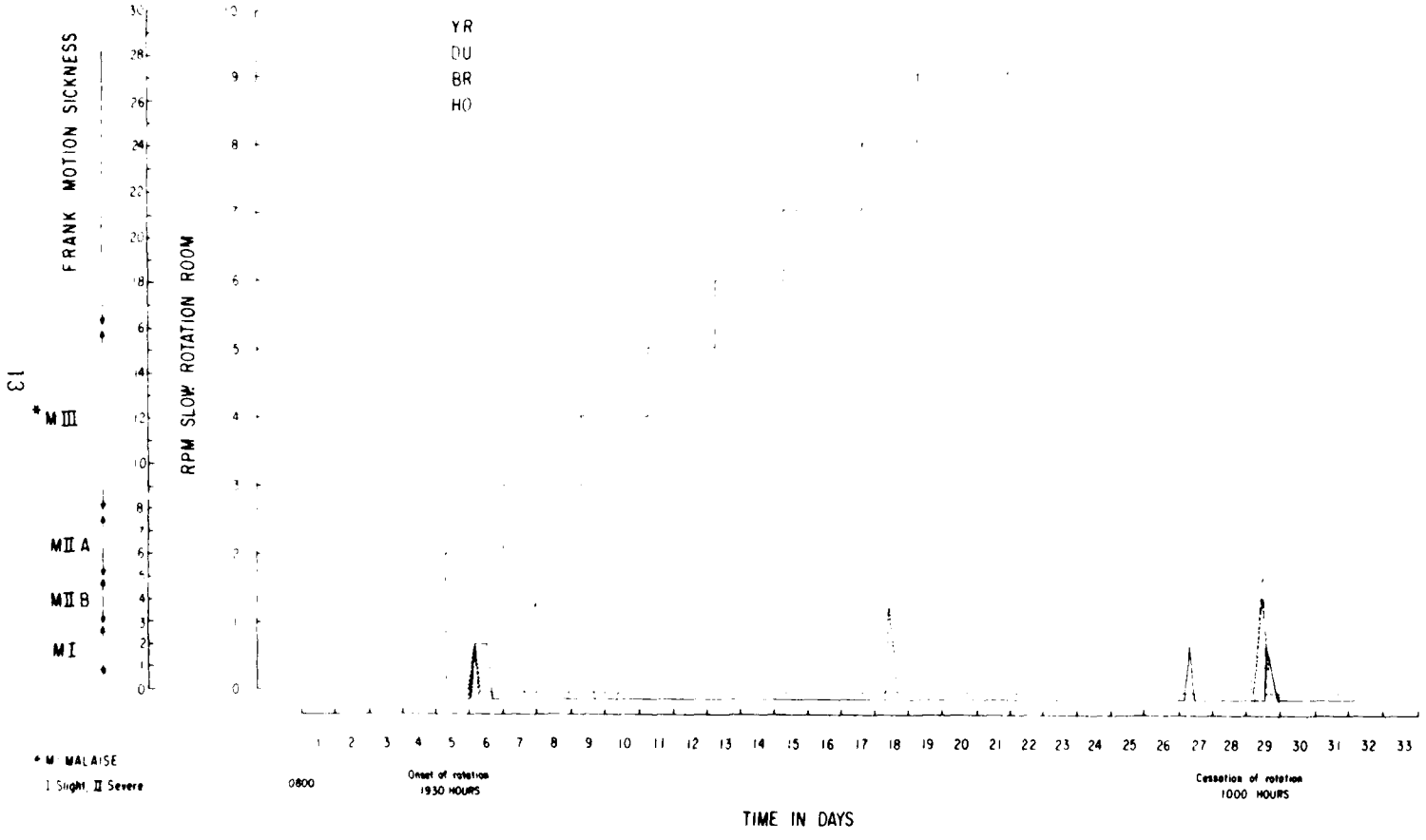

Figure 5

The stress profile in the SRR and changes in level of motion sickness symptoms in four healthy subjects exposed to rotation over a period of nearly 25 days.

The findings just described pointed the way to speed up the adaptation process by substituting "standardized head motions" for general activities at each incremental increase in velocity; fatigue would become the limiting factor in making continual head motions (H-M's), and adaptation would take place in a relatively short period. Two experiments were conducted (12), and the stress profile of the second one, along with a summary of the findings, is shown in Figure 6. The duration of rotation was a little over two days, during which the subjects made 1000 H-M's at each one-unit increase in velocity, except at 10.0 rpm when 500 were made and were followed by generalized activities. These sessions occupied about four to five hours of each day, and there were no restrictions on the subjects' moving about at other times. In the perrotation period one of the subjects did not manifest any overt V-II symptoms; in one the symptoms were trivial, and in the third they were slight. On cessation of rotation the subjects maintained the same rankings with regard to V-II symptoms but all were ataxic.

The demonstration that V-II manifestations could be prevented by incremental exposure to otherwise highly stressful Coriolis accelerations not only reflects the great capacity of the V-I system to effect homeostatic adjustment, but also underlines the fact that adaptation must take place during exposure at 1.0 and 2.0 rpm when even overt V-I manifestations are not expected. The phenomenon of over-adaptation was again demonstrated, and the curious finding noted that on sudden return to the stationary environment, V-II symptoms were either absent or, with a single exception, trivial. Ataxia was severe and aggravated by head motions, indicating V-I contribution to the response.

Brief Exposures Using Physiological Sensors. It is unfortunate that so little attention has been given to the earliest appearance and disappearance of V-II symptoms along a time axis. Only in this way will we gain some notion regarding latency, and whether we are dealing with first-, second-, or even third-order symptoms or their complications. Although our experimental program has not been specifically directed to this end, a few heretofore unpublished observations have been made, in collaboration with Dr. S. C. Dunn, which are relevant.

The shortest latency we have observed was in the onset of sweating which occurred while a susceptible subject flexed his head toward the shoulder and back to the upright (Figure 7). The event recorder was activated by the experimenter and indicated that the first head movement began about 3 to 4 seconds* prior to upstrokes from the baseline, registered by a recording hygrometer (upper curve) and polygraph (GSR) (lower). The top curve measured the alterations in moisture content of a dry gas led through a capsule with the open side applied to the palmar surface, and thence to a recording hygrometer. The delay time between capsule and hygrometer recordings was about three

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*These measurements were made from the original record.

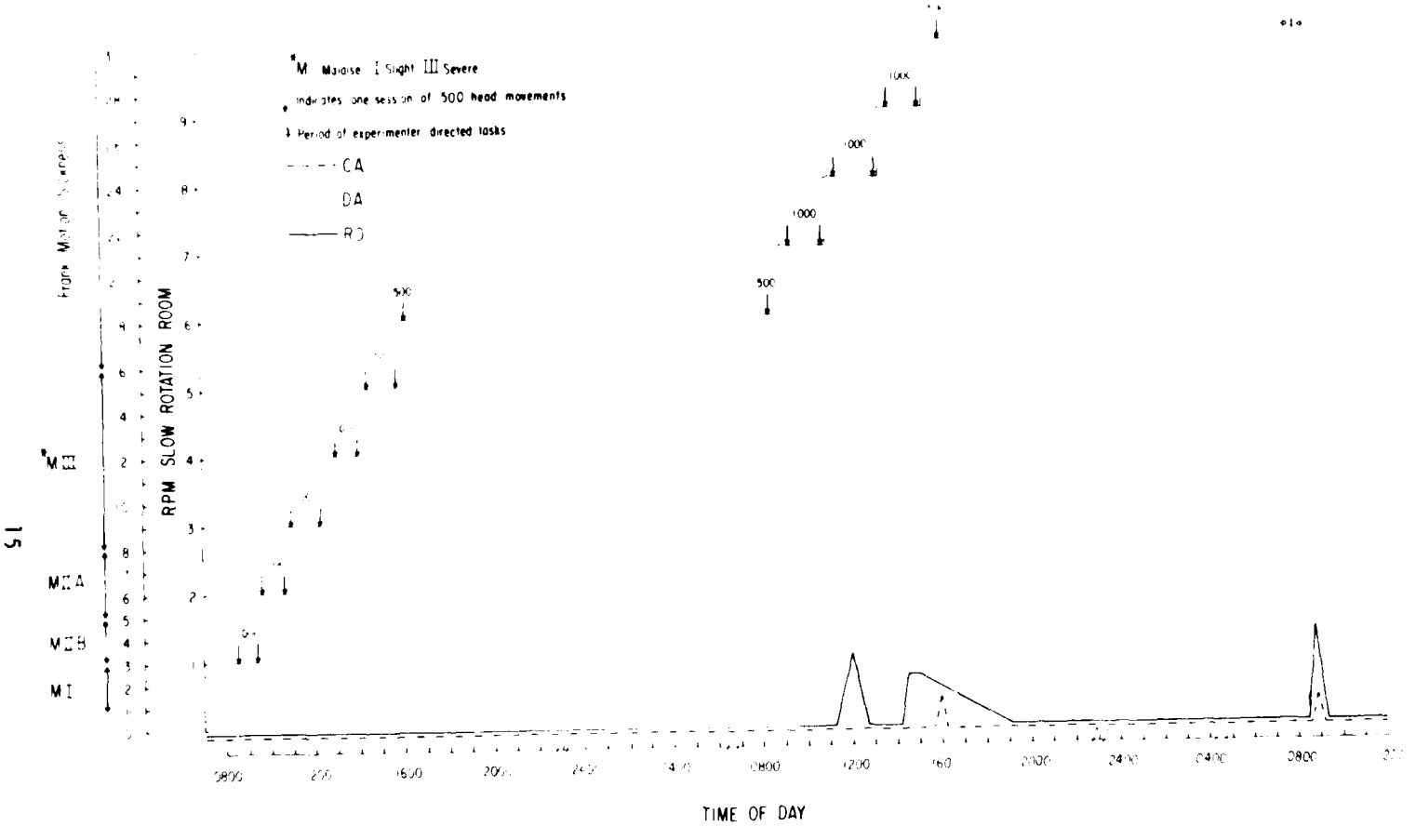


Figure 6

The stress profile in the SRR and manifestations of motion sickness in three healthy subjects exposed to rotation for about two days. The large number of head motions accounted for the rapid adaptation.

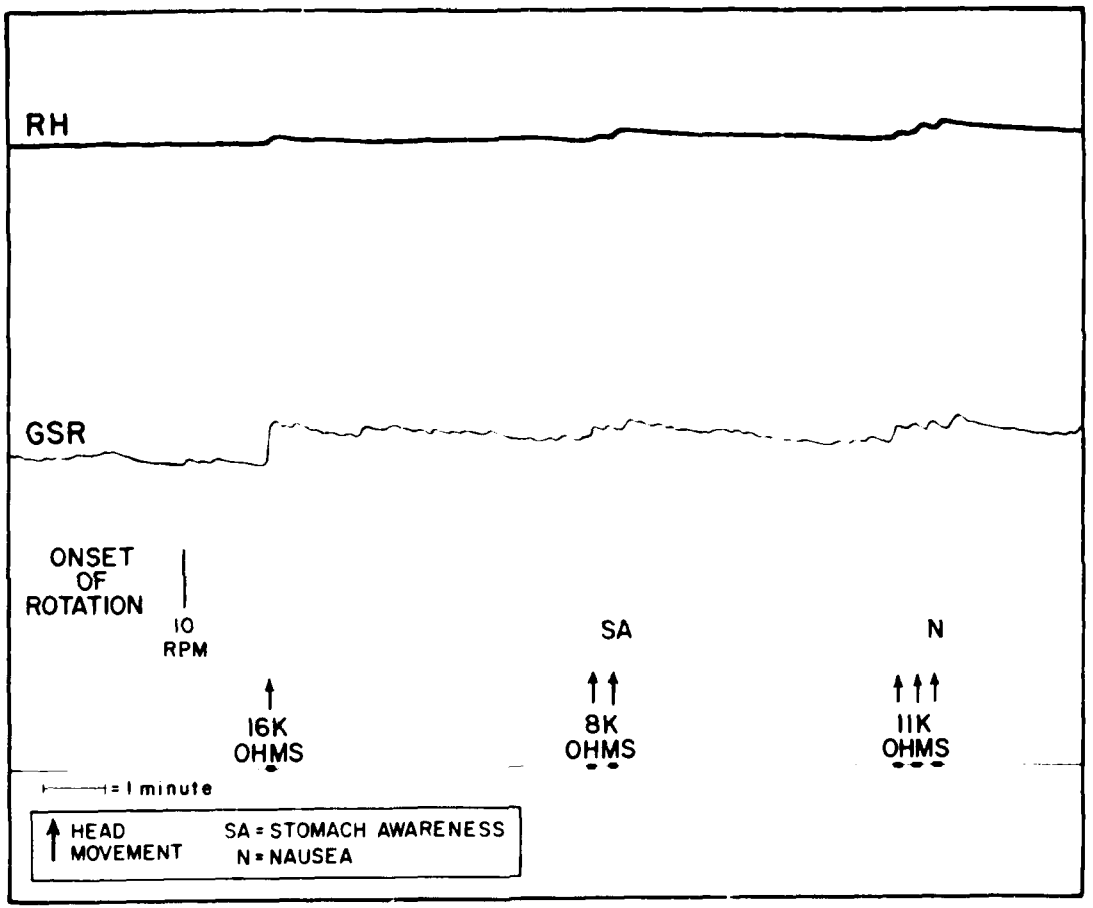


Figure 7

Record showing changes in percentage relative humidity (top curve) of a dry gas passing through a capsule, with open side toward the palmar surface, and changes in GSR (second curve) while making six head flexions toward shoulder at times indicated by event marker (bottom line).

seconds. It is noteworthy that the upstroke of the two curves occurred almost simultaneously and that the upper curve slowly rose and then fell but not quite to the baseline. The upstrokes on both curves occurred within a second of the onset of the vestibular stimulus. The failure of the upper curve to return to the baseline is probably significant in the light of subsequent events. A little more than four minutes later, two head movements were made, with identifiable rises on both curves, and the subject experienced sweating and epigastric awareness soon after. Three head movements were made a little less than four minutes later and slight nausea was experienced. Note the slow return of the upper curve toward the baseline.

These findings suggest that in a susceptible person, stronger stimulation of the vestibular system is reflected almost immediately by a V-II manifestation, implying responses from a normally independent system. The short latency is consonant with irradiation of vestibular activity to the site of origin of the autonomic response. The long perseveration after the stressor was off suggests the release of a chemical factor or prolonged reverberating activity.

In Figure 8 are hygrometer and temperature recordings from another susceptible subject. The temperature was recorded from the tympanic membrane by the method of Benzinger (4) and reflected near body-core temperature. The curves were traced from the original record, and the phenomenon of temporal summation to repeated stimuli is clearly indicated. The upper curve showing changes in moisture content of the gas passing through the capsule indicates that sweating first occurred about the time that slight epigastric distress was experienced and that, without further vestibular stimulation, there was an increase in sweating associated with nausea. Meanwhile, finger and core temperatures declined slightly but significantly. Finally, after two more head movements there was nausea, severe sweating, vomiting, and an accelerated fall in finger and core temperature. This may be an instance of an avalanche effect, with secondary influences playing a large role comparable to what has been described as a "massive discharge of the sympathoadrenal system."

A schema depicting the major events underlying the systems cross-over manifestations in these two cases is shown in Figure 9. It is almost self-explanatory. In the first case, the assumption of an initiating reflex with release of a neurohormone at the effector site to account for long perseveration is plausible at least. The underlying events in the second case are less clear; but, it must be assumed that temporal summation of the stimulus was necessary initially and that the severe subsequent events in response to only four additional head motions must have involved a self-energizing mechanism which may have occurred through the "secondary influence" pathways. In the discussion to follow the point will be made that certain factors may, in the absence of the V-II factor, be primary but in its presence have a greater effect than the V-II factor.

Measurement of Susceptibility to SRR Sickness. Thousands of observations have been made in connection with establishing criteria for the clinical evaluation of different levels of severity of motion sickness (16) and with the human assay of

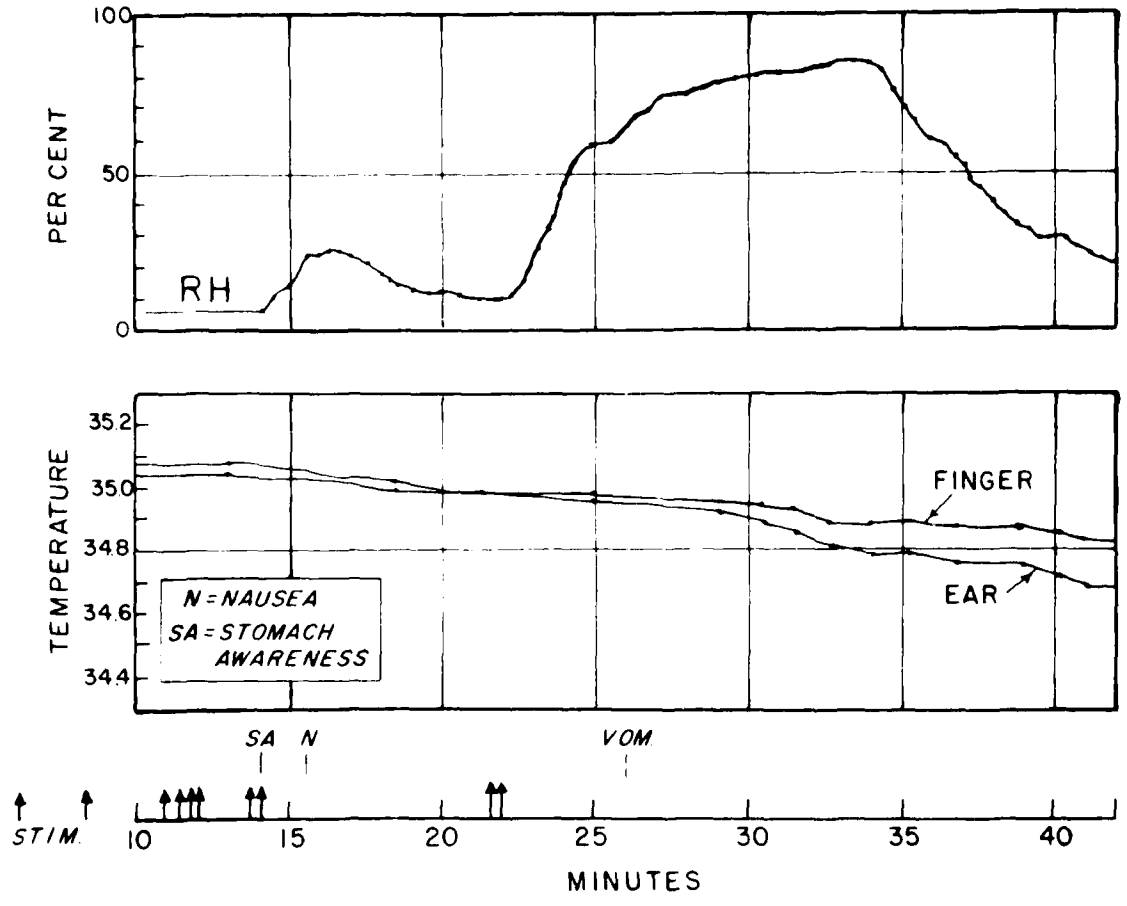


Figure 8

Changes in relative humidity (upper curve) and in body-core (ear) and finger temperature of a susceptible person exposed to Coriolis accelerations generated by head motions in a room rotating at 10 rpm.

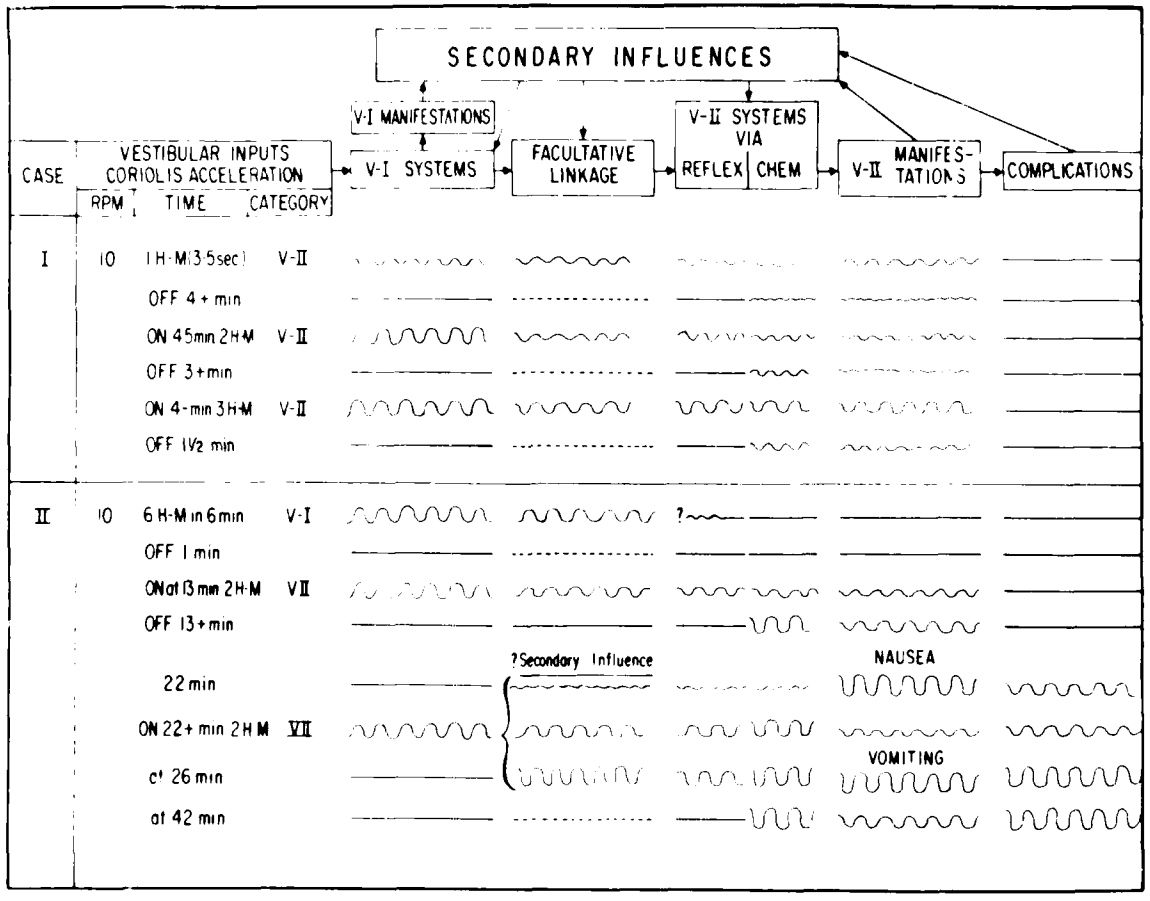


Figure 9

Schema depicting possible events and processes underlying V-II manifestations in two highly susceptible persons (see text). (Explanation of lines to be found in legend of Figure 1.)

antimotion sickness drugs (50). An important object with regard to clinical diagnostic criteria was to establish reliable "end points" which would be far less severe than frank motion sickness. This posed a requirement to choose a level of stimulation which would evoke mild symptoms after the seated subject had completed 50 to 100 head movements (about 5-10 minutes). A stronger stimulus would produce an "avalanche effect," with the symptoms out of the experimenter's control; and a weaker stimulus either might not evoke symptoms after 300 head movements, the cutoff point, or evoke symptoms below the level of severity required.

Angular velocities up to 20.0 rpm, the maximum permitted in the "old" SRR, were used. Even at 20.0 rpm subjects with very low susceptibility might not meet the desired "end point" after 300 H-M's; this reflects the smaller stress, using limited planes and arcs of rotation, compared with unlimited motions associated with generalized activities.

The severity of the symptoms was graded (Table I) along a continuum scored in points (1 to 50) and broken down into four levels below 16 points, with "frank sickness" at or above this point (16). Moderate Malaise (M II A: 5-7 points) and severe Malaise (M III: 8-15 points) were demonstrably reliable end points. When the exposure time to reach the end point was very brief, reflecting an error in estimating a person's susceptibility from information obtained by questionnaire, the end point was overrun. Invariably, however, the symptoms quickly disappeared unless vomiting occurred, in which event the subject's susceptibility remained preternaturally high for the remainder of that day. This residual effect could not have been due to a continued disturbance in the vestibular system, as demonstrated in connection with V-I disturbances, but must have involved mechanisms either underlying temporal perseveration in V-II manifestations but at the subclinical level, or an augmentation of secondary etiological factors, thus decreasing the level of primary stressful accelerations required to evoke symptoms.

When the same subject was exposed to various levels of stress, the order of appearance as well as the severity of the symptoms was changed. This was also seen when subjects differing in susceptibility were exposed to the same level of stress. It must be kept in mind that only selected symptoms were used in estimating the severity of V-II symptoms.

The probable mechanisms underlying the V-II manifestations evoked while making "standardized head motions" are depicted in Figure 10. The subject, seated, listened to a recording that indicated the duration and the direction of rotation of the head away from and return to the upright. The stressor effect could be indicated by time or the number of head motions which involved time.

At low angular velocities, and taking into account individual susceptibility, standardized head rotations out of the plane of the room's rotation do not evoke overt V-II manifestations even though long continued. At 10.0 rpm, ten discrete head motions in a relatively insusceptible subject evoke strong V-I manifestations but no overt V-II manifestations. The possibility that this stimulus may predispose the subject

Table I

Diagnostic Categorization of Different Levels of Severity of Acute Motion Sickness

Category	Pathognomonic 16 points	Major 8 points	Minor 4 points	Minimal 2 points	AQS* 1 point
Nausea syndrome	Vomiting or retching	Nausea+ II, III	Nausea I	Epigastric discomfort	Epigastric awareness
Skin color		Pallor III	Pallor II	Pallor I	Flushing/Subjective warmth ≥ II
Cold sweating		III	II	I	
Increased salivation		III	II	I	
Drowsiness		III	II	I	
Pain					Headache
Central nervous system					Dizziness
					Eyes closed ≥ II
					Eyes open III

Levels of Severity Identified by Total Points Scored

Frank Sickness	Severe Malaise	Moderate Malaise A	Moderate Malaise B	Slight Malaise
(S)	(M III)	(M IIA)	(M IIB)	(M I)
≥ 16 points	8 - 15 points	5 - 7 points	3 - 4 points	1 - 2 points

*AQS = Additional qualifying symptoms. +III = severe or marked, II = moderate, I = slight.

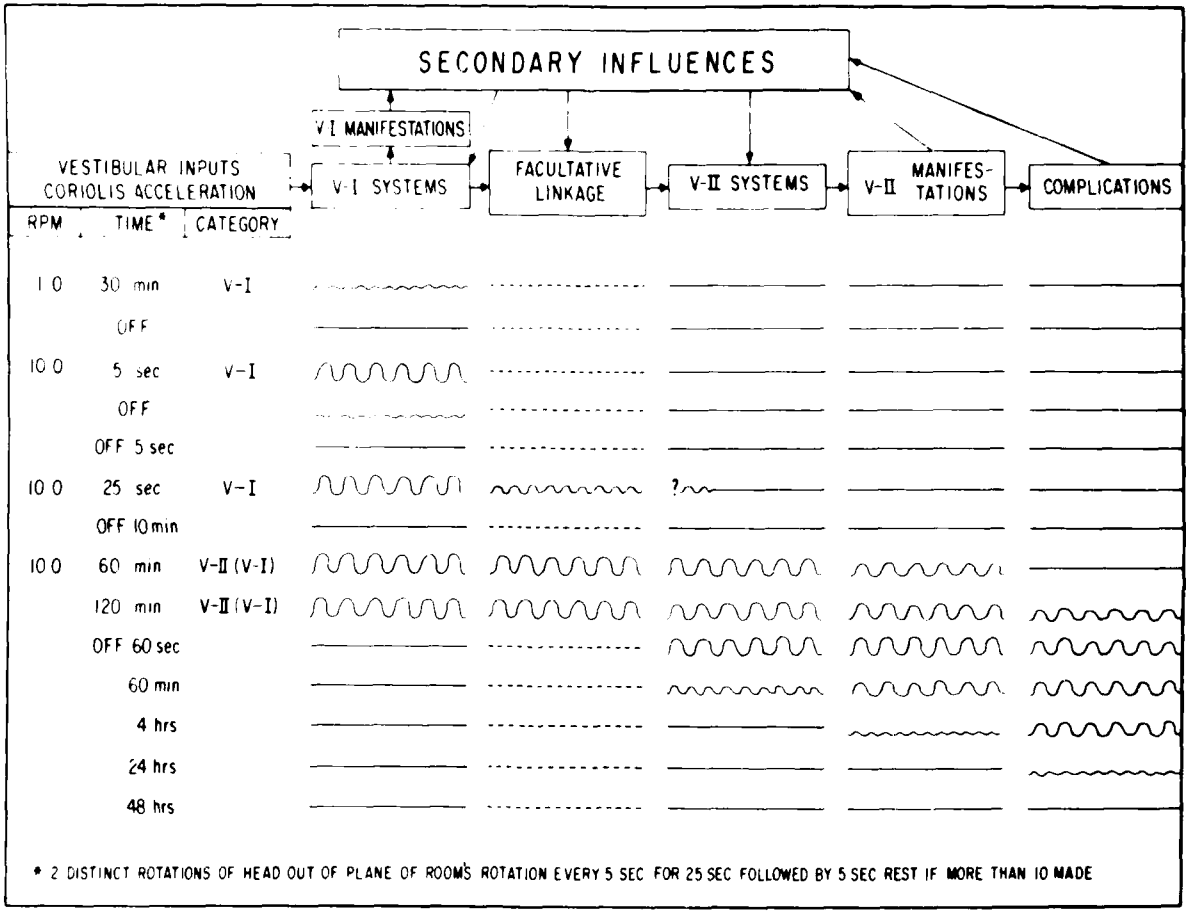


Figure 10

Schema depicting possible events and processes underlying manifestations evoked by Coriolis accelerations acting on the non-acoustic labyrinth. (See Figure 1 for explanation of lines.)

to any subsequent stimulation may or may not be demonstrable and is indicated in the schema (Figure 10) by a partial irradiation of the stimulus through networks where the facultative linkages take place. A strong stimulus leads to severe symptoms and complications, with relatively slow return to the control homeostatic state.

Prolonged Exposure with Sudden Change in Body Orientation with Reference to the Axis of Rotation During the Perrotation Period. A unique feature of this experiment was the provision for subjects to walk and carry out their tasks while horizontal with respect to the Earth vertical (17). This was made possible by the use of air-bearing supports and custom-fitted articulated fiberglass molds (Figure 11). Four subjects participated in two different experiments involving adaptation to the force environment with the room rotating at 4.0 rpm. One pair of subjects, initially in the horizontal mode, was changed to the vertical mode at the end of two or three days when symptoms of motion sickness had disappeared; in the second experiment they began in the vertical mode (Figure 12). The order was reversed for the second pair. When in the horizontal mode, the subjects spent approximately 6 hours of a 24-hour day in the air-bearing device, 6 to 10 minutes upright and the remainder of the time recumbent on a bunk.

The principal object of the experiment was to compare susceptibility to motion sickness in the two modes and to determine whether adaptation in one mode transferred to the other. The findings indicated no significant difference in susceptibility in the two modes and that transfer of adaptation was excellent. Moreover, after cessation of rotation symptoms usually disappeared quickly.

A byproduct of the experiment demonstrated important differences between motion sickness and postural disequilibrium during adaptation to the rotating, and subsequent to the stationary, environment. In the start-horizontal mode, adaptation ensuring freedom from symptoms of motion sickness on change to the vertical mode did not prevent ataxia. In the start-vertical mode, the adaptation resulted in a great decrease in ataxia; this adaptation perseverated throughout the finish-horizontal mode and as long as 36 hours afterward. This implied that the dynamic processes underlying postural homeostasis involved muscular activities largely rendered static when subjects were in the horizontal mode.

DISCUSSION

COMPARISON OF V-II WITH V-I MANIFESTATIONS

The cardinal V-II symptoms are related to V-I manifestations only through the contributions made by vestibular organs in initiating the system cross-over responses and in initiating their disappearance through removal of this primary stimulus by means of adaptation. In this way V-I and V-II symptoms are "associated" although otherwise they are manifestations of disturbances in different systems.

Indeed, the V-I and V-II symptoms are so different that, with rare exceptions, they are distinguishable when coexistent; the former are what might be expected as a

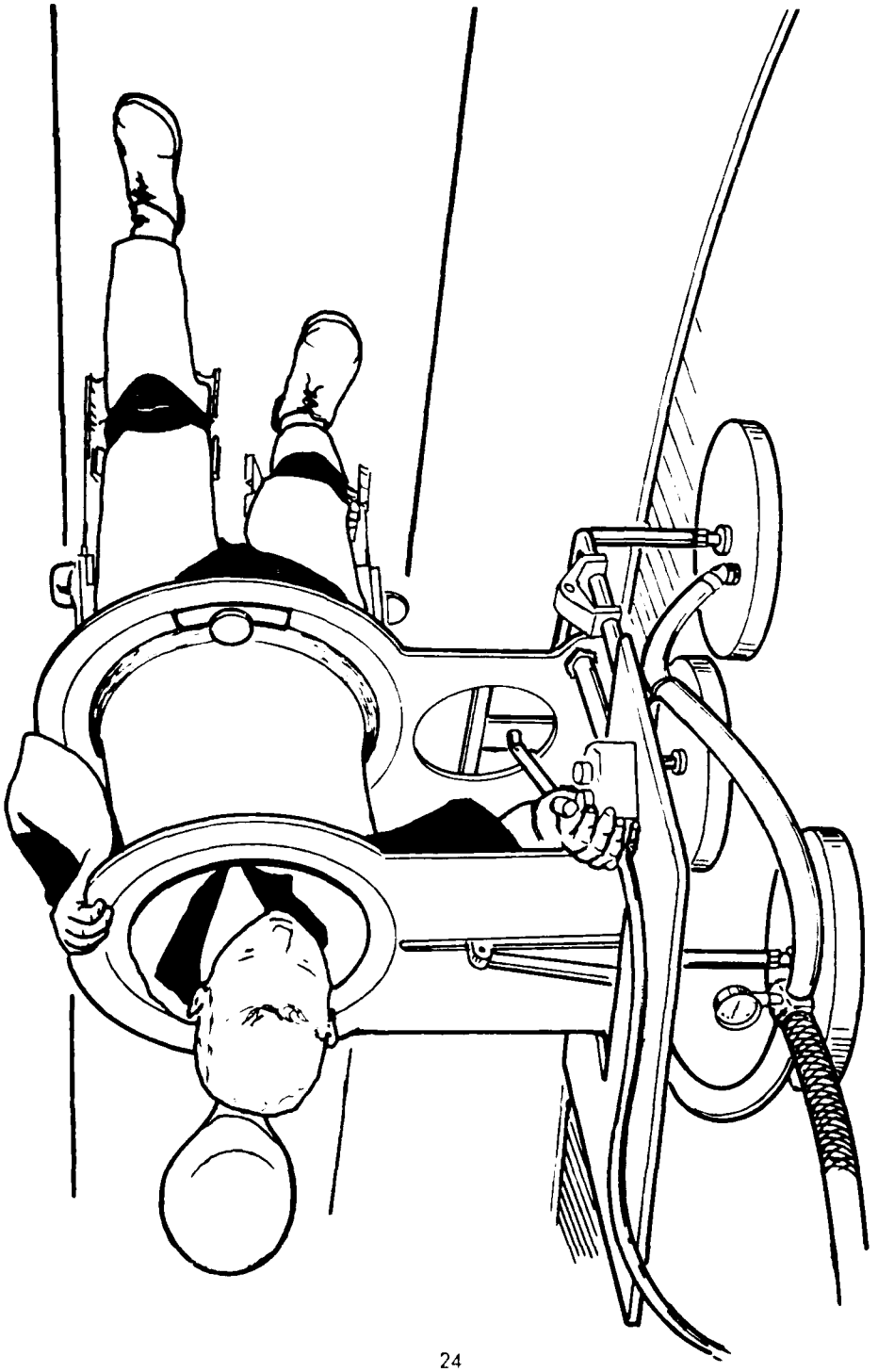
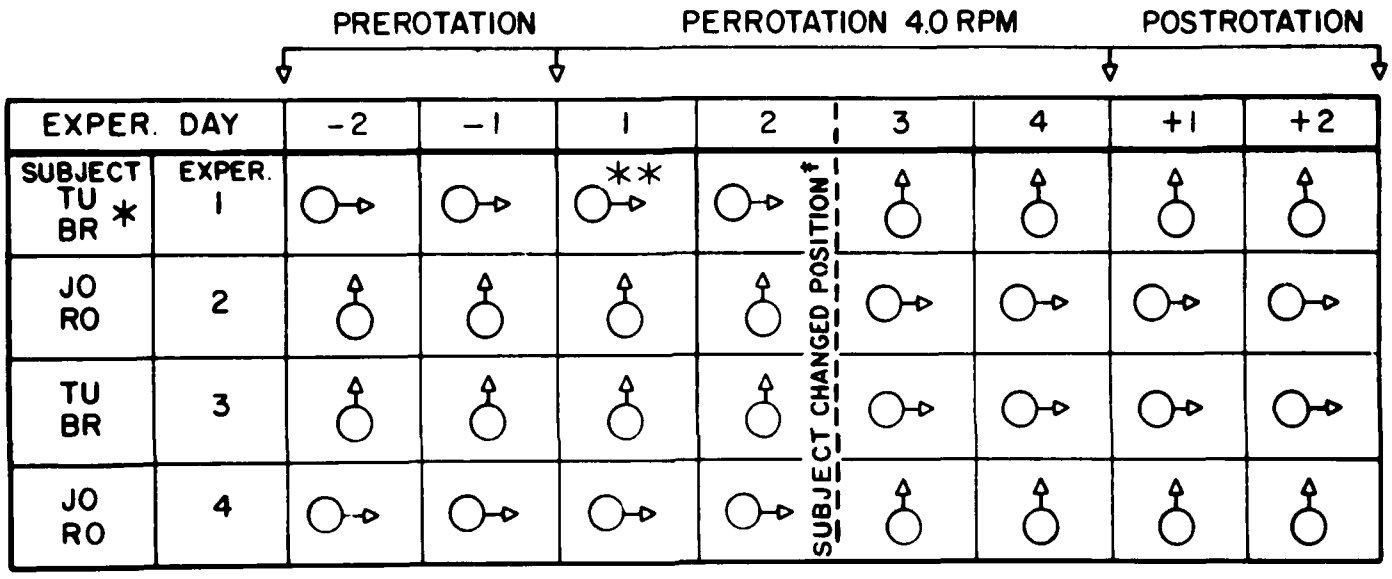


Figure 11

Subject walking in air-bearing device. Long axis of body at right angles to axis of room's rotation.



* TU and BR spent 3 days in horizontal mode during perrotation phase in first experiment.

** When in the horizontal mode, during a 24-hour period subjects spent between 5 ½ and 6 ½ hours in the air-bearing device, between 6 and 10 minutes upright, and the remaining time on a bunk.

† Occurred around 0930 hours.

Figure 12

Showing changes in orientation of four healthy subjects throughout an experiment in the SRR. Not sudden changes in the perrotation period. See text.

result of disturbances in the vestibular system while the latter are not. In comparing the symptomatology it is convenient to make a distinction between the symptoms per se and abstract phenomena derived from their study, which reflect or implicate underlying events and processes.

There are only a few easily studied overt V-I manifestations in the SRR. Gernandt (7) has emphasized that the threshold of somatic outflow is about three times as high as that of visceral nerve responses, which implies that one is missing opportunities to use the more sensitive indicators. Most investigators have centered their attention on disturbances of eye motions with their consequences of visual illusions and possibly blurred vision and dizziness.

If V-I manifestations are meager, the reverse is true for V-II symptoms which are difficult to study except under systematically, carefully controlled conditions. Some of the initial symptoms certainly involve the visceral nervous system and include sweating, flushing, and pallor due to vasomotor activity, drowsiness, a decrease or increase in salivation, and a host of symptoms generally referable to the gastrointestinal tract although of central origin. The nausea syndrome is but one of many symptom complexes, although rightfully regarded as the most important because of its distressing and incapacitating features. Second- and third-order symptoms are evoked presumably via hypothalamic motoneurone activity releasing the pituitary hormones ADH and ACTH.

With regard to some of the derived phenomena, V-I and V-II manifestations are, as might be predicted, either very different, similar, or identical. Typically, short-latency, maximal initial manifestation and short perseveration after the stressor is off characterize V-I manifestations while the opposite is true for V-II symptoms. Although there is evidence of secondary influences affecting both, they are less prominent in V-I than in V-II manifestations. Adaptation is highly characteristic of both although, as will be noted below, there are interesting differences. During prolonged exposure to rotation, there is a gradual response decline involving both V-I and V-II manifestations; however, only a few systematic observations have been made (6,24,43), and these usually have been concerned either with V-I or V-II symptoms. Postrotation symptomatology has not been studied in detail. A holistic approach involving the total symptomatology in a single experiment is needed.

The phenomenon of transfer of adaptation has been studied in connection with both V-I and V-II manifestations (23), and there are many similarities of a qualitative nature between the two. Collins (37), Guedry (20), and others (27) have studied intensively the "habituation of nystagmus" and the conditions under which it does and does not "transfer."

THE FACULTATIVE LINKAGE

The evidence is irrefutable that the initial V-II symptomatology represents responses to the irradiation of vestibular activity either to unusual sites or to sites

stimulated in an unusual way. It is assumed that this is made possible by the bizarre nature of the vestibular input affecting facilitory-inhibitory mechanisms. Particularly germane to this discussion are the recent findings of Gernandt (8) which demonstrated functional differences between the medial longitudinal fasciculus (MLF) and the reticular formation (RF) in conducting vestibular impulses to the oculomotor nuclei. Above a critical frequency of electrical stimulation of the vestibular nerve in cats, the RF would no longer transmit vestibulo-ocular impulses, while high-frequency stimulation via the MLF evoked strong contractions of the extraocular muscles. Moreover, it was demonstrated that only the vestibulo-ocular impulses traveling through the RF were under the inhibitory control of extraocular muscle-stretch activation. Gernandt's findings are analogous to a "facultative linkage." They are a clear demonstration of vestibular impulses, within certain frequency constraints, using a pathway through the reticular formation and demonstrably "open" to modulating influences of an inhibitory nature.

The fact that one head motion can result in a measurable increase in sweating, with a short latency, suggests that this crossover may be due to vestibular activity going directly to the cell assembly whence the symptom had its orgin. There is much additional evidence bearing on this last point. Thus, a high degree of selectivity in evoking symptoms by regulating the strength and duration of the stimulus is demonstrable; drowsiness, for example, may be evoked in the absence of other overt symptoms, either as an initial manifestation or after other symptoms have disappeared through adaptation. Again, the orderly appearance of one symptom after another over a relatively long period suggests that discrete cell assemblies are stimulated one after another. The relatively rapid disappearance of mild V-II manifestations when central commands pre-empt common pathways again suggests a predominantly reflex phenomenon.

When the V-II stressor is on for a long period before symptoms appear (temporal summation) or if there is a massive V-II response with long perseveration of symptoms, the participation of transmitter or other chemical agents, or both, must be considered. What is more in doubt, is whether chemical agents are released at sites of origin of initial V-II symptoms or released elsewhere and transported to sites of origin; both may be operant. Our findings in this connection have contributed little except to pique our interest. One of my associates, Mr. Colehour, conducted an experimental probe and found substantial increases in serotonin and histamine in the blood of dogs made sick with Compound 48/80, but no trace of either was found in the cerebrospinal fluid. The relevant literature is too voluminous to review here, but the findings of Pappenheimer (41), finally proving that cerebrospinal fluid from a sleep-deprived animal results in drowsiness when injected into another animal, are of great interest to us inasmuch as drowsiness, in the absence of other overt symptoms, may be produced by a properly controlled vestibular stimulus.

SECONDARY INFLUENCES

One constraint in this report was to avoid, insofar as possible, details in connection with the role of secondary influences on V-I and V-II manifestations. Some comment cannot be avoided, however, in presenting even the simplest preliminary

conceptual scheme. The reasons include 1) the need to emphasize that a secondary influence in the presence of a vestibular precipitating factor may be a primary factor in its absence; e.g., vision in the absence of vestibular stimulation may evoke symptoms similar to V-II manifestations; 2) the need to point out the possibility that similar symptoms evoked with and without cognitive participation may involve different mechanisms; and 3) the very important role of pathological factors.

In the SRR it is very easy to evoke V-II manifestations with the subject unaware that he is being stressed, simply because proprioception may remain below the level of awareness and seldom qualifies as a true sensation. One important point is that there may be no awareness of "conflict." On the other hand, strong sensations, e.g., pain or smell, and either phenomena or abstractions registering dread, fright, or horror, and physiological inputs which provide conflicting cues, may all evoke symptoms similar to V-II manifestations. The last example is particularly pertinent in that one person may readily resolve the conflict while another may become first confused, then "sick." This could be an example of a V-I manifestation leading to mental confusion or "conflict" and subsequently evoking M-II symptoms. On the other hand, L-D subjects are characteristically confused (10) and in conflict to a far greater degree than normal subjects over discrepant information from optic and proprioceptive sources and they do not become ill. Just as a "dragging" sensation on the abdominal viscera may seem to evoke nausea in a normal person, the same feeling in an L-D subject is, at worst, a mild discomfort, pointing to the difference between associative and etiologic connotations.

We have observed striking instances when a naive subject on his first exposure in the SRR manifests far more severe symptoms than on subsequent occasions even when exposed to stronger Coriolis accelerations, and this is best explained on the basis of secondary influences. An illness such as a respiratory infection, which otherwise would not result in nausea and vomiting, may greatly increase susceptibility to V-II symptoms. In subjects during long exposures in the SRR we have observed a dramatic change in symptomatology as the result of psychological disturbances. One difference between conditions in the SRR and those during acrobatic flying may be that the latter, by causing distraction or anxiety, decreases or increases, respectively, susceptibility to V-II symptoms.

THE CONCEPT OF FUNCTIONAL VESTIBULAR RESERVE

Henderson's concept of a functional reserve represents an extension of Cannon's concept of homeostasis. An attempt will be made to demonstrate that under the standardized conditions in the SRR, it is possible to distinguish clearly between the events and processes when the vestibular functional reserve is not exceeded and those wherein there is a "failure" of homeostasis in vestibular mechanisms, allowing "nonhomeostatic" (V-II) events to occur.

In Figure 13 is shown a fairly realistic stress profile, with the shaded areas indicating the limits of functional vestibular reserve and the unshaded areas indicating failure. Not shown on the graph is the time-course of changes following cessation of

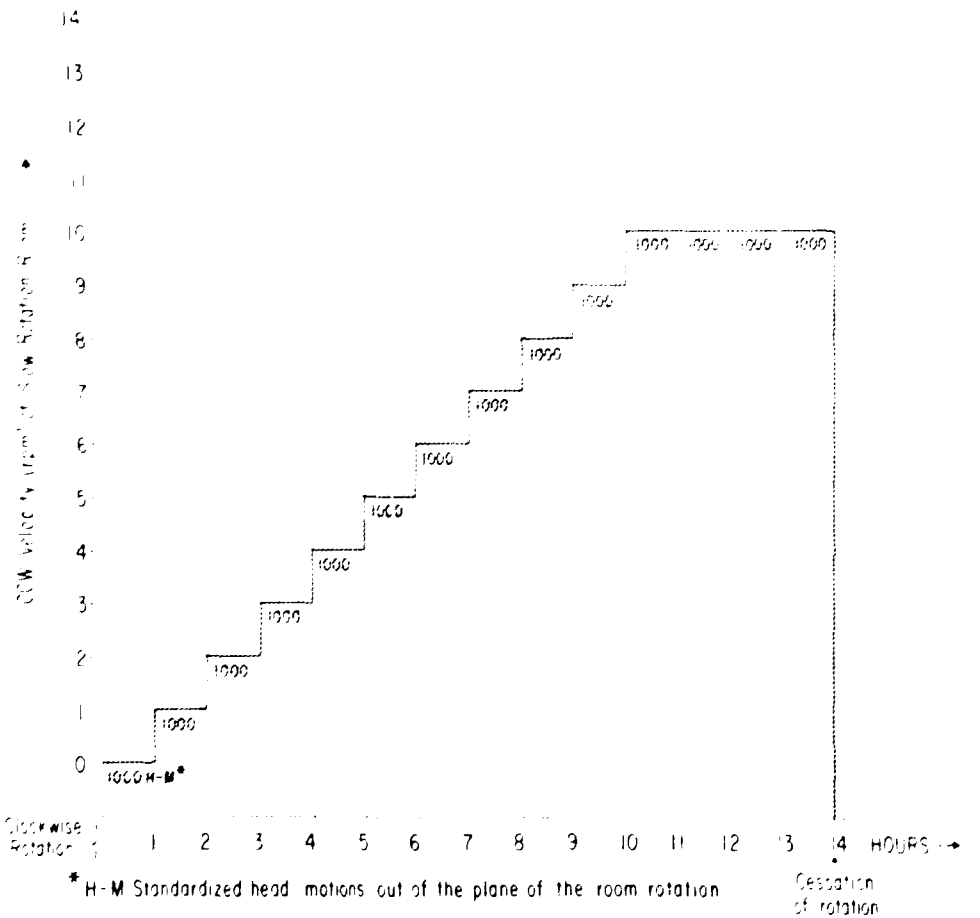


Figure 13

Predicted changes in functional vestibular reserve with reference to motion sickness susceptibility during incremental increases to stressful Coriolis accelerations in the SPR (see text). Insusceptibility indicated by shaded area and susceptibility by open spaces.

rotation and on rotating in the clockwise direction because our data are meager. The discussion, however, will include these two dimensions.

In the case of the fictitious subject used as an example, his functional reserve before counterclockwise rotation began is shown by the shaded area extending up to 7.0 rpm, and there is no reason to suspect that it would be significantly different if rotation were in the clockwise direction. After making 1000 H-M's out of the plane of the room's rotation at 1.0 rpm, his functional reserve has been raised to "normal" or control level at 2.0 rpm; thereafter, with the same exposure at velocities up to and including 10.0 rpm, the same incremental increase in reserve is shown, although at 10.0 rpm the shaded areas are clipped at the top. Note that with each additional 1000 H-M's at terminal velocity, the functional reserve is increased. In Figure 13 this is shown at the bottom along the zero velocity band. Thus, if after completing the head motions at 3.0 rpm, the subject were brought back to the stationary environment, mild V-II manifestations would be experienced which would not have been the case at lower velocities. After he completed the head motions at 5.0 rpm, the V-II manifestations, on return to the stationary environment, would have reached their upper level of severity. This remains unchanged until overadaptation at 10.0 rpm brings about a decline and, finally, immunity to V-II symptoms on cessation of rotation. That this increase applies to velocities higher than 10.0 rpm is simply an extrapolation of what has occurred previously, but the curious feature, although our information is not fully sufficient on this point, is that overadaptation increases the functional reserve on return to the stationary environment.

The curves in Figure 14 illustrate individual differences in degree of adaptation on making the same number of H-M's at unit increases in angular velocity. In all instances the functional vestibular reserve (FVR) is shown to be a function of the number of head movements made per unit step. These curves are consonant with our experimental findings that some persons demonstrate a relatively large increase (solid lines) and others a small increase (broken lines) on making the same number of H-M's. The concept of a functional vestibular reserve is applicable generally; in the SRR with H-M's standardized it could be expressed as a functional reserve velocity.

A series of systematic experiments specifically directed toward determining the precise change in functional vestibular reserve by having the subject continue to "overadapt" at a given or terminal rpm has just been initiated by my associate Dr. Reason. A not inconsiderable body of information indicates that, after a large number of head motions are made at terminal velocity, V-II symptoms are absent on return to the stationary environment, although V-I symptoms and ataxia are manifested.

Loss of adaptation on return to the stationary environment occurs rapidly at first, then more slowly, so that at the end of four days' rotation, much of the reserve has been lost. Systematic studies along these lines are in progress.

An experimental probe was conducted by my associate Dr. Cramer, requiring two subjects to make 600 H-M's at each unit increase in counterclockwise velocity,

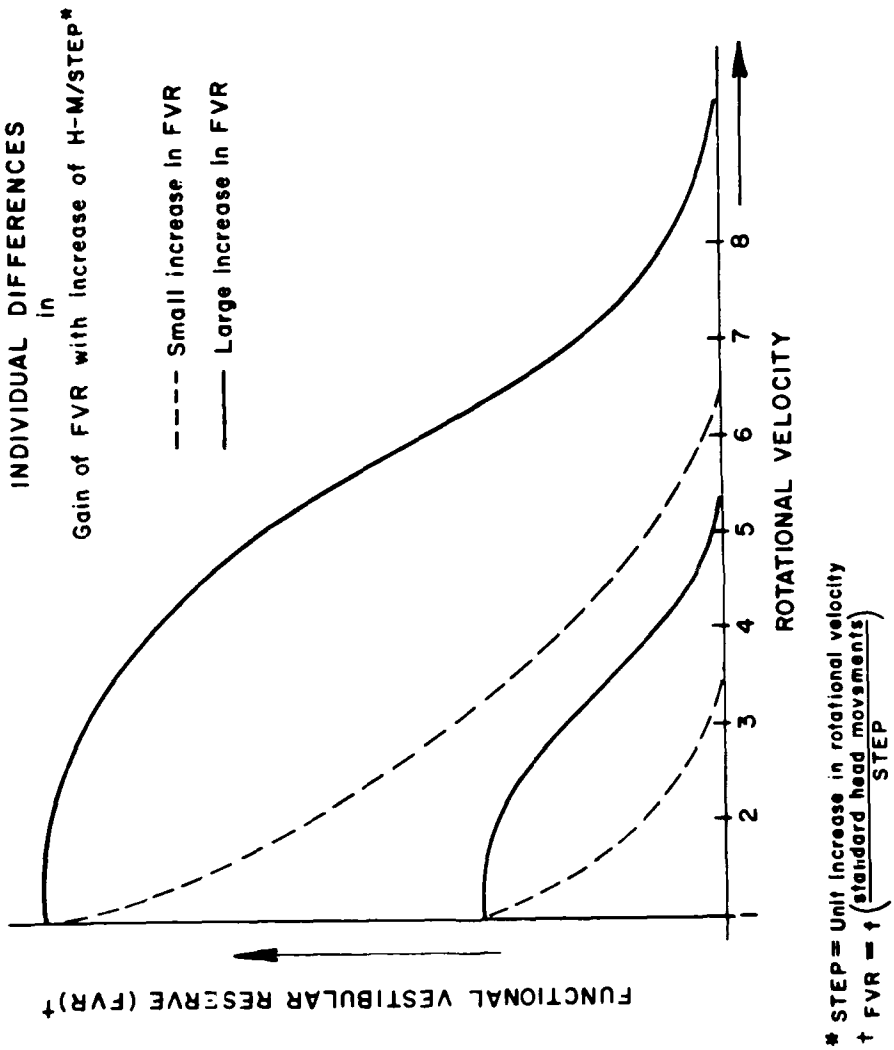


Figure 14

Individual differences in gain in functional vestibular reserve with increases in number of standard head movements made at each unit increase in rotational velocity.

up to and including 5.0 rpm,* and then exposing them to a clockwise velocity of 5.0 rpm. There were no V-II manifestations at the terminal velocity when rotating counterclockwise, but mild (M II A) manifestations were experienced by Subject A after 20 H-M's and Subject B after 30 H-M's when rotating clockwise. These findings imply that the new integrative pattern is direction specific.

From the theoretical standpoint, it is curious that overadaptation at a terminal velocity seems to favor a reduction or even immunity to V-II symptoms on return to zero velocity rather than the reverse. This suggests a general suppressing effect, yet, that it must be direction specific. The direction specificity would implicate Groen's hypothesis (19), while suppression would seem to implicate efferent vestibular activity. These implications are tentative and await further study.

HOMEOSTATIC MECHANISMS

At least three different types of homeostatic mechanisms are involved in the symptoms manifested in the SRR. The strictly vestibular V-I manifestations represent a disturbance in the vestibular and its normally articulating systems, and their appearance and disappearance are closely related to stressor-on and stressor-off. With regard to the Coriolis oculogyral illusion and nystagmus, there is evidence of a functional reserve in that they are not observed or experienced when the head is flexed to one shoulder with the SRR rotating at 1.0 rpm. At higher velocities, however, the normal integrative patterns of the central nervous system no longer serve, and symptoms are a measure of this inadequacy. Hundreds of head movements may be required to establish a new pattern, resulting in a new homeostatic state. This new state is highly specific if head movements are limited to flexions of the head to one shoulder, although there is some transfer beyond the precise plane in which the head is moved with reference to the body and the precise room velocity at which the homeostatic adjustment takes place. The nature of the adjustment involved is, presumably, a compensatory mechanism of opposite sign. So long as the head is fixed, the pattern does not change, implying its static character in the absence of changing sensory inputs. The new state does not seem to carry a penalty in terms of performance decrement, although there may be an upper limit in terms of velocity of the room at which homeostatic adjustment would be satisfactory. Although precise experiments have not been made to detect performance decrements in other sensory or sensorimotor modalities after complete adaptation at 10.0 rpm, there was no evidence for this in such measurements as were made. If this is the case, it would appear that only the V-I systems are involved. How long this pattern would "hold" on cessation of rotation with head remaining fixed is not known, but there is no evidence of a spontaneous return to the normal or initial state during periods of sleep aboard the SRR, or for shorter periods after cessation of rotation when conditions

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*Subject A made only 300 H-M's at 4 and 5 rpm

are better for detecting a change. On the other hand, head motions speed up the process of restoration on return to a stationary environment.

The homeostatic mechanisms involving V-II manifestations involve both V-I and V-II systems. A "failure of homeostatic processes" in the vestibular system best characterizes its inadequacy to prevent vestibular influences irradiating to sites other than those with which it articulates under normal conditions, or even under slightly abnormal conditions. The latter possibility is in accord with the evidence that adaptation in the vestibular system can occur at 1.0 rpm without any evidence of V-II manifestations. Presumably the homeostatic mechanisms involved here are the same as those described above under V-I manifestations. This, however, accounts only for the primary or initiating stimulus, and the far more difficult problem must be faced with regard to the vast array of V-II symptoms after the V-II stressor is off. Even if we limit the problem to initial symptoms, this is difficult for at least two reasons. First, the V-II manifestation was not in response to a physiologic need. Second, stimulation at the effector site of origin may be abnormal. In the case of cold sweating the cooling effect might well act to reduce the sweating through normal dynamic homeostatic processes which spontaneously seek to establish a stable state. On the other hand, drowsiness either persists long after the V-II stressor is off or in response to a weak level of V-II stimulation, and the homeostatic mechanisms involved in restoration of a "normal" alert state are unknown.

ATAXIA

The problems of ataxia have little direct connection with those of motion sickness, and such problems are different in a rotating spacecraft generating a centripetal force of 0.165 g, on the Moon (0.165 g), and in a slow rotation room with the same angular velocity as the spacecraft. On the Moon the platform is stable, and the lunar standard of gravity affects the otolith apparatus which, in turn, must alter its tonic modulating influences, including canalicular contributions. In a rotating spacecraft generating 1/6-g unit, the Coriolis accelerations on walking would lead to postural disequilibrium which would not completely disappear through mechanisms of adaptation, and such movements which would affect the level of centripetal force would act to increase or, to a small extent, decrease postural stability. For the above reasons, any simulator on Earth has limitations for simulating walking on the Moon or in a rotating spacecraft at lunar or other subgravity levels of centripetal force. Nevertheless, some of the difficulties can be partially overcome, and the findings in the SRR can be extrapolated in most respects to force environments in ships on turbulent seas and, if the person walks about, to turbulence in aircraft.

Inasmuch as here our attention is on motion sickness, further discussion will be limited to pointing out the characteristic nature of the nonvestibular homeostatic mechanisms involved.

The evidence suggests that with the onset of rotation, there is a change in visual-postural coordination, resulting in a rearrangment on the basis of movement-produced

visual feedback (reafference) (26, 28) to ensure adjustment or compensation. This would account for the lack of readaptation to the stationary environment while, for example, the subjects remained recumbent in body molds or on their bunks for periods measured in days. Moreover, this was not greatly affected by the fact that, by means of limited head movements, they readapted to the stationary environment insofar as the vestibular organs were concerned. Walking was required to abolish the errors of opposite sign which appeared when they began to walk in the stationary environment. Experimenters have learned that the nonvestibular component of postrotation ataxia is soon abolished by running, while omnidirectional movements of the head abolish the vestibular component. The investigator can manipulate the experimental design so as to disassociate completely ataxia and V-II manifestations of which motion sickness is a part. The comparison between cessation of rotation in the SRR and debarking after a voyage is obvious. The reason ataxia is absent or minimal after deplaning is that little walking is done aloft.

MOTION SICKNESS

Motion sickness is one of several terms which has been used to indicate a constellation of functional disturbances causally related to force environments in some means of conveyance or in some other device (29,40,47). It was introduced in 1881 by Irwin (29), a clinician, and gradually gained wide acceptance because it met the test of "convenience" by its etiologic and symptomatic connotations. In view of the difficulty in devising perfect terms, motion sickness is likely to remain as a useful designation for a very long time, certainly in common parlance and probably in medical discourse. The limitations of the term arise from the fact that it has never been defined in a precise manner, although there is general agreement that nausea and vomiting are cardinal symptoms, and in most of the experimental studies of motion sickness in man (1,3,30,31,33-36,38,45) and in nearly all of the experimental work on animals (2,39, 42,45,46,49) vomiting has been used as the diagnostic criterion or "end point."

In writing the present report the shortcomings of motion sickness as a designator were insurmountable. This dilemma was resolved by introducing more precise terms, but never leaving the reader in doubt as to where "motion sickness" fitted into the conceptual scheme. It would seem reasonable to suggest that all system cross-over manifestations be included under the designation motion sickness and that a distinction be made between them and symptoms associated with motion sickness, which might be termed V-I symptoms. At the very least, this would place no restrictions on studies as they now are being carried out, yet would offer the clear benefits to be derived from making important distinctions between the two categories of manifestations. This does not, however, resolve the need for a term to indicate all instances of system cross-over manifestations primarily of vestibular origin and, indeed, all functional central nervous system disturbances with similar manifestations, whatever the primary etiologic factor.

Throughout this report it was necessary time and again to point out gaps in our knowledge concerning almost every significant aspect of motion sickness. The present

approach not only reveals the deficiencies in our knowledge concerning motion sickness from the operational standpoint, but also indicates the possibilities for extending the investigation of central nervous system mechanisms, using controlled artificial vestibular stimulation as an instrument.

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STRUCTURAL ELEMENTS IN THE CONCEPT OF MOTION SICKNESS

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Ashton Graybiel

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A slow rotation room in a laboratory environment provides an excellent instrument for the study of motion sickness because the experimenter can control not only the stressful Coriolis accelerations, but also other important procedural and environmental variables. By exploiting this control, combined with the judicious selection of experimental subjects, it was possible to confirm many previous findings and demonstrate that manifestations of disturbances in the vestibular system fall into two distinct categories. In the first category are reflex phenomena evoked by Coriolis accelerations when the head is rotated out of the plane of the room's rotation, and revealed through systems which, under natural stimulus conditions, have functional articulations with vestibular receiving areas. The symptomatology in the second category comprises an epiphenomenon superimposed on any manifestation of the first, when the unusual vestibular activity, presumably through facilitory-inhibitory processes, irradiates to cells or cell assemblies not normally stimulated.

Selected experimental findings are used in defining the characteristics of manifestations in the two categories and in demonstrating the nature of the facultative linkage between the otherwise independent systems underlying manifestations in the two categories. It will be shown that the experimenter, by manipulating mainly vestibular homeostatic mechanisms, can prevent the appearance of manifestations in the second category, control their severity when evoked, and lose control only when these symptoms are relatively severe or perseverate long after the stressful accelerations have ceased. Practical and theoretical implications are discussed, including the concept of "functional vestibular reserve."

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KEY WORDS	LINK A		LINK B		LINK C	
	ROLE	WT	ROLE	WT	ROLE	WT
Motion sickness						
Acceleration effects						
Homeostatic mechanisms						
Vestibular apparatus						
Illusions						
Nystagmus						
Ataxia						
Vertigo						
Space medicine						